Periodontal disease is an inflammatory disease, involving plaque production through several bacteria, that affects our global population health. Individuals with extreme periodontal disease face a gradual increase in tooth loss. Recent research has displayed the possible molecular pathway for how the inflammatory disease causes the acceleration of alveolar bone loss through the NLRP3 (NLR family pyrin domain containing 3) inflammasome. Here I review recent studies that illuminate the NLRP3 pathway and its contribution to periodontal disease. Researchers have found the activation of the NLRP3 pathway is influenced through the presence of functioning mitochondria and K+ efflux, but not dependent upon their roles. Once the inflammasome is produced, it proceeds to activate caspase-1, caspase-4, and caspase-5. These caspases lead to production of IL-1β, which causes the differentiation of osteoclasts and the breakdown of bone. Over time, the breakdown of bone from osteoclasts can lead to the loss in teeth. Tranilast, MCC950, and Urolithin A are inhibitors of NLRP3 and can be potentially used in periodontal patients to slow down the effects of alveolar bone resorption. From the information gathered, the in vitro evidence found in the articles in this review paper leads to strong molecular evidence that the NLRP3 inflammasome and its subsequent bone loss is one of the many causes of periodontal disease.